evidently occurs in the remains of the ureter above the calculi, and when the pus is discharged into the bladder a condition of things is produced almost as bad as when the kidney was still in work.

THE PRESYSTOLIC APEX MURMUR OF AORTIC REGURGITATION: WITH NOTES OF A WELL-MARKED EXAMPLE.

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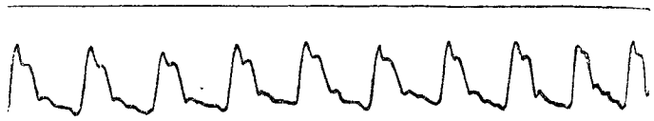
A LOW-PITCHED presystolic murmur may occasionally be heard over a small area immediately around the impulse in cases of aortic regurgitation. The high-pitched, blowing, diastolic murmur is possibly audible from the base to the apex and even outwards into the axilla, but just at the point of the heart's impulse a rumbling sound takes its place. Such a low-pitched sound will probably be made to disappear by slight pressure of the stethoscope, and may thus be overlooked. Cases, however, have been recorded by Austin Flint, Guiteras, Gairdner, Osler, Maguire, Lees, and others in which a more or less well-marked presystolic apex murmur was present where aortic valve disease was the only heart lesion. The following case is an example of that curious occurrence of the murmur. In this instance the presystolic bruit was not low-pitched and rumbling, but of that loud, rolling character that reminds one of the sound produced by a flapping sail as it is filled by a puff of wind. Well marked though the murmur was, the mitral orifice proved to be of natural size.

CASE 1.—A man thirty-two years of age was admitted into the Bristol General Hospital on Aug. 28th, 1894, under the care of Dr. George Parker, who has kindly given me permission to publish the case. The patient was a thin, wiry, rather short man. He was markedly cyanosed, and although there was no orthopnœa he was in great distress. He generally lay on the right side with his head resting upon his right forearm, but quickly rose to the sitting posture at the onset of a frequent short cough. There was slight œdema of the legs, but no swelling of the abdomen. The history was interesting, but need not be given here; it may, however, be mentioned that he apparently had not had rheumatism, and there was no evidence of syphilis. The heart's impulse was in the fifth space in the nipple line, where it was accompanied by a distinct presystolic thrill. The cardiac dulness was very slightly increased on the left, but was well marked at the fourth and fifth spaces to the right of the sternum, extending about two inches outwards in the latter space. There was a loud presystolic apex murmur of rolling character, which increased in intensity as it approached an abrupt termination. Although, however, the termination was abrupt and loud the first sound was not distinct, and the notes state: "There is nothing that can be called a first sound; what appears to be the first sound has a broken character." The area over which the murmur was well marked was small and situated immediately around the impulse, but a duller rolling sound was heard more widely. In addition to this murmur a *bruit de galop* was heard in a somewhat unusual position below and outside the impulse. Three separate sounds were there distinctly audible. There was no systolic murmur at the impulse, but between that point and the sternum a high-pitched systolic bruit became audible, and was heard from the fifth to the second right and left spaces. A diastolic murmur of high-pitched blowing character was audible down the left of the sternum. Behind the sternum another murmur, loud and half squeaking, half musical, was sometimes heard. This the patient said that he had noticed. The pulse was regular, 100 small, but aortic in character (see Chart). The patient steadily grew worse and died on Sept. 24th. Nothing fresh of importance developed except moderate effusion into the right pleura, which was twice aspirated. On the day of death it was noticed that a systolic murmur was audible at the impulse, and that the triple sound formerly heard outside the apex had disappeared.

Necropsy.—At the post-mortem examination the situation of the apex was found to be at the sixth rib in the nipple line, and the right auricle extended three inches to the right of the sternum. The pericardium was healthy. The heart was enlarged and weighed twenty-two and a half ounces, the enlargement being mainly in the left ventricle, the wall of which measured from three-quarters to seven-eighths of an inch in diameter. There was comparatively little dilatation. The mitral valves were perfectly healthy, and the orifice of normal size. It measured four inches. The tricuspid valves were also healthy. The orifice measured four inches and a half. The main feature of interest was in the aorta. Immediately above the valves the vessel was surrounded by a raised area varying in breadth from three-quarters to one inch and a half. The upper border was serpiginous and the surface irregular and pouched. The post-mortem examination did not take place until forty-four hours after death, and the thickened area was deeply blood-stained, but seen earlier it would no doubt



Pulse tracing.

have presented the grey appearance of the aortitis generally attributed to syphilis. The aortic valves were slightly thickened, but only one, the right posterior, was deformed, and that was retroverted. The endocardium over the septum ventriculorum was greatly thickened where the regurgitant stream had impinged. The orifices of the coronary arteries were not obstructed and the vessels themselves were quite healthy. The heart muscle looked normal and the portion microscopically examined showed no pathological changes. Microscopic sections of the affected aorta gave evidence of active disease. There was fibroid thickening of the greater part of the intima and media, but groups of leucocytes were scattered through the outer half of the vessel and through its sheath.

Remarks.—In this case a well-marked presystolic murmur and a presystolic thrill existed in association with incompetence of the aortic valves, but with a perfectly normal mitral orifice. It is now well known that the physical signs of mitral stenosis may occasionally be present in cases of disease of the aortic orifice; and in examining the records of a large number of cases of death from various forms of heart lesion at Guy's Hospital I discovered several more or less well-marked instances of a presystolic apex murmur where the aortic orifice alone was affected. The following single example may be briefly referred to by kind permission of Dr. Hale White.

CASE 2.—A man forty-five years of age was admitted into Guy's Hospital under the care of Dr. Hale White in 1892. The heart's impulse was in the sixth space, three inches outside the nipple line. There was a presystolic thrill at the apex, and the notes state: "There is a harsh presystolic murmur at the point of impulse in the sixth space. At the base there is a to-and-fro murmur." The pulse was "splashing," being 92 to the minute.

Necropsy.—At the post-mortem examination the mitral orifice was found to measure four inches and a half, and the valves were quite normal. The aortic valves were also quite healthy; but there was pouching of the sinuses of Valsalva, with dilatation of the first part of the aorta, which led to regurgitation through the aortic orifice. There was a thickened patch of endocardium on the septum ventriculorum where the regurgitant stream had impinged. Dr. E. W. Goodall, then medical registrar, adds a note at the end of the clinical report. He says: "As regards the presystolic thrill and bruit, they were marked, and mitral stenosis was diagnosed as existing. The thrill and bruit may perhaps be accounted for by Austin Flint's hypothesis."

Remarks.—There is one point of morbid anatomy I should like to draw attention to in this case and in the one that precedes it—that is, the situation of thickened endocardium upon the septum ventriculorum showing the point of impact of the regurgitant stream. This is of some interest since it negatives the explanation Dr. Sansom has suggested for the presystolic murmur of aortic regurgitation. He has thought it probable that the regurgitant stream, impinging upon the anterior mitral flap, may set up vibrations that lead to the production of the presystolic sound.

These two cases show that the regurgitant stream need not be directed upon the valve, and one other case that may be briefly noticed seems to show that a presystolic murmur may exist in association with aortic valve disease without any regurgitation being present.

CASE 3.—A man aged forty-seven years was twice admitted to Guy's Hospital under the care of Dr. Pavy in 1879, and on both occasions a presystolic apex murmur was noted by different clinical clerks. The medical registrar also heads the report: "Well-marked presystolic bruit." At the post-mortem examination, however, the mitral orifice was found to admit four fingers. The aortic valves were adherent half way up their edges, but were considered by Dr. Goodhart to have been competent. Allowing Dr. Goodhart's conclusion to be correct, it is obvious that not only need the regurgitant stream not impinge upon the anterior flap of the mitral valve, but that no regurgitant stream need be present for the production of a presystolic apex murmur.

This leads us to a form of presystolic murmur in which I am somewhat interested, a murmur occurring in dilatation of the heart in which neither mitral stenosis nor aortic disease is present, but the most common lesion is adherent pericardium. A paper upon this subject was read by me at the last meeting of the British Medical Association. A copy of this paper sent for abstract to a foreign journal fell into the hands of the conductors of the *Medical Press and Circular*, and was published by them without my knowledge in the number for Oct. 10th, 1894. That being the case, I must refer any interested reader to that periodical for detailed information. Brief notes of twelve cases of diastolic or presystolic apex murmur occurring without mitral stenosis or disease of the aortic valves are given. Ten of them are from the Guy's Hospital records. Of the twelve cases, in eight the pericardium was universally adherent, and in only two the pericardium was healthy. In five other cases of general adhesion of the pericardium a presystolic apex murmur was heard, but they have been excluded because the pathologist thought the aortic valves probably incompetent. The existence of a presystolic murmur in the above cases was noted not only by the clinical clerks but also in the majority of instances by the physician or medical registrar. Thus, Dr. Goodhart adds marginal notes in two instances. In one, a girl aged eleven years in whom the mitral orifice was found to measure four inches, he describes "a distinct presystolic thrill and a rumbling, long, diastolic murmur in the fifth and sixth spaces." In another case, a boy aged nine years, where the orifice measured four inches and a half, Dr. Goodhart mentions "a short, roaring, diastolic sound," and calls it "a diastolic mitral." Dr. Frederick Taylor also noted a large diastolic thrill and a diastolic rumbling sound where the mitral orifice was perfectly normal. Dr. Pye-Smith and Dr. Hale White both diagnosed mitral stenosis incorrectly from the presence of a presystolic murmur, and Dr. Newton Pitt also noted a presystolic apex murmur where the mitral orifice measured four inches and seven-eighths.

In all these cases there was adhesion of the pericardium. It is therefore evident that there are two entirely different pathological conditions that will give signs simulating those of mitral stenosis—namely, disease of the aortic orifice and adherent pericardium. Such being the case, it is reasonable to conclude that some condition common to both must be concerned in the production of the murmur. The most obvious feature is dilatation of the left ventricle, and it may be supposed that the mitral orifice is small compared with the size of that cavity and thus produces a virtual stenosis. The large size of the orifice, however, in some of the cases of adherent pericardium excludes the possibility of such an explanation, and curiously enough it was in those forms of heart disease in which a virtual stenosis probably exists that, with one exception, I failed to find instances of a presystolic apex murmur having been noted. I refer to cardiac dilatation due to chronic Bright's disease, and to less definite but probably not infrequent causes such as alcohol and overwork. Since we reject a virtual stenosis as an explanation we must consider what other conditions may be present in a dilated left ventricle. It may have been noticed in the post-mortem room that when a dilated ventricle is present the large anterior flap of the mitral valve may be held taut by columnæ carneæ and chordæ tendineæ that have not fully shared in the dilatation. During life, as the ventricle becomes filled during diastole this state of tension must be present in the large mitral flap, which, held out in the moving blood currents instead of falling against the septum, may be left free to vibrate. Dr. Stacey Wilson in his interesting lecture upon Dilatation of the Right Side of the Heart gives a similar explanation for the diastolic murmur sometimes heard over the right ventricle.[1] On endeavouring to accept such a view we, however, again meet with difficulty. If such an explanation were correct one would expect a presystolic apex murmur to be common in dilatation of the heart from any cause and especially in those cases of dilated heart in which the mitral valves remain healthy—for example, those consequent upon Bright's disease and other causes affecting the heart muscle. In the Guy's Hospital records for twenty years one instance only, however, was discovered in cases of that nature, and that, curiously enough, was a well-marked example. It was in a man aged forty-six, admitted three times, and on every occasion mitral stenosis was diagnosed. The last admission was under Dr. Pavy in 1879. At the post-mortem examination a large heart was found but no definite cause for hypertrophy discovered. The mitral orifice admitted three fingers. The absence of mitral stenosis struck the medical registrar—I think Dr. Mahomed—who heads the clinical report: "Presystolic bruit heard in this case, but no stenosis found." Although a presystolic murmur was noted in the above case of cardiac dilatation in which neither adherent pericardium nor disease of the aortic orifice existed, we are obliged to admit that it is a remarkable exception, and that there is virtually no evidence to prove that simple dilatation of the heart will give rise to a presystolic murmur. That being the case, we are driven to consider whether there is anything else in common between enlarged hearts due to disease of the aortic orifice and to adherent pericardium. Possibly in both the innervation may be affected. In the pericarditis that precedes adhesion of the pericardium it is difficult to understand how the nerves coursing superficially over the bloodvessels can entirely escape, and in cases where aortic regurgitation is due to aortitis, causing dilatation of the aortic orifice, the nerves passing downward in the sheath of the aorta may possibly be affected, as Lanceraux has long believed. In Case 1, for example, leucocytes scattered through the sheath of the aorta were often aggregated around the nerves, and although it was not evident that the nerves had materially suffered, toxines may have been present and have caused functional disturbance. In cases, however, where the rumbling apex murmur is associated with disease of the aortic valves and not of the aorta we can hardly suppose that any affection of the nerves is present, yet the frequency of cardiac pain suggests that in some obscure way the innervation of the heart is affected or that the heart muscle is functionally disturbed. The first case in which I heard a presystolic murmur that was proved to have been unassociated with mitral stenosis, the cardiac failure was of the nature that seems to me to point to disturbed innervation. In a boy aged nine, without œdema or liver enlargement, there was orthopnœa, great distress, and a rapidly acting heart beating with sufficient force to cause visible vibration of the whole body. On recently looking again at the microscopic sections of the heart muscle pathological features previously overlooked were noticed—viz., some proliferation of the endothelial cells of the pericardium and groups of leucocytes in the subserous tissue. In other words, the boy appears to have died in a very early stage of pericarditis. Since there is no evidence of myocarditis we may suppose that death occurred from functional disturbance of the nerves of the heart muscle or by toxines. Allowing that in some cases of presystolic murmur occurring without mitral stenosis there is a deranged nervous mechanism or some unusual alteration in muscular action, the question of the mode of production of the murmur still remains to be explained. Possibly there is a loss of muscular tone and vibrations of the muscle wall are set up by the inrush of blood on contraction of the auricle. But whatever the explanation of the murmur may be the fact remains that mitral stenosis is not the only heart lesion that will give rise to a presystolic murmur.

In closing, reference may be made to a case that Dr. Perry has kindly given me permission to mention. This case presents an additional feature of interest in that it bore resemblance to some cases of mitral stenosis in another particular. Of every four cardiac beats only two were felt at the wrist. The case was a woman aged twenty who was admitted under the care of Dr. Wooldridge

[1] THE LANCET, Sept. 15th, 1894.

in August, 1888. She had been seen by him as an out-patient, when a well-marked presystolic murmur and a soft systolic murmur were present at the apex. After admission, however, the presystolic murmur seems to have disappeared and a diastolic murmur was audible between the nipple and the sternum. In August, 1892, the patient was again admitted under the care of Dr. Perry. Over the mitral area a "rough rumbling presystolic murmur" was then heard, as well as one systolic and more blowing in character. A presystolic thrill was also present. As already mentioned, it was thought that only two pulsations were felt at the wrist to every four cardiac beats, and sphygmographic tracings seemed to support that view. The patient died.

The clinical clerk gives an abstract of the result of the post-mortem examination, after which he somewhat cautiously remarks: "The diagnosis of mitral constriction that was fairly confidently made in the ward was thus proved to be apparently a mistake." The heart was a peculiar one; it was large, weighing eighteen ounces. The apex was formed mainly by the right ventricle, which was greatly hypertrophied, the muscle wall measuring three-quarters of an inch in diameter. The tricuspid orifice measured five inches and the mitral three inches and three-quarters. The aortic valves were healthy, but the pulmonary had a few small vegetations upon them. The most curious feature was the marked atheroma of the pulmonary artery, which extended into its small branches. Apparently the hypertrophy and dilatation of the right ventricle were due to obstruction in this vessel.

The case affords fresh evidence of the possibility of the existence of a presystolic murmur apart from mitral stenosis, but apparently does not throw much light upon the production of such a murmur. In all probability the murmur was produced in the right ventricle, and the great thickness of the wall is worthy of note—a feature one would think must have been in some way connected with the production of the abnormal sound. Common though a dilated right ventricle is, a rumbling sound sufficiently loud to attract the attention of most observers is rarely heard in association with such dilatation. A previous suggestion has, however, been made—namely, that disturbed innervation enters into the etiology of these anomalous murmurs. Possibly the curious and extensive atheroma present in the pulmonary artery in this case followed an arteritis that had affected all the coats of the vessel and its sheath, leading to injury of some nerves of the cardiac plexus.

Since writing the above Dr. Audeoud of Geneva has kindly sent me his paper upon "Le Rétrécissement Mitral Fonctionnel." He has noticed diastolic and presystolic apex murmurs which have disappeared. Dr. Audeoud speaks of a functional stenosis which he considers to be due to temporary spasm of the mitral orifice. The results of necropsies in two cases are given. One of the cases resembles one under Dr. Perry above mentioned. In a man aged sixty-two in whom a diastolic apex murmur had been present nothing was found but a large heart. The mitral orifice admitted three fingers, and the valves were healthy.

NOTE ON "THE SURGICAL SIGNIFICANCE OF SMALL CALIBRE RIFLES."

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THREE very valuable and interesting articles on the above subject have recently appeared in the *United Service Gazette*.[1] They are based on a paper by Lieutenant H. R. Stiles, Assistant Surgeon, United States Army, which appeared recently in the journal of the Military Service Institution, New York, and presumably the views put forward in them are those held by that officer.

In the first article Lieutenant Stiles' opinion of the cause of the occurrence of those frightfully lacerated and destructive injuries produced by rifle bullets at short ranges, to which the term "explosive effects" is commonly applied, is stated. He considers that they are to be explained by what has been called by Continental surgeons the "hydraulic theory." Upholders of this theory of the production of these extensive injuries look upon a limb or a part of the body as a sealed vessel containing liquid, or an almost liquid substance, and consider that when it is penetrated by a projectile possessed of high velocity, the liquids of the tissues produce within it similar effects to those seen in a tin can filled with water or a moist substance, and sealed, under like circumstances. In the latter case the bullet produces, besides the entrance and exit holes, numerous ruptures in the sides and in the top and bottom of the vessel quite unconnected with the actual perforations due to the bullet itself. The edges of these rents in the metal are turned outwards, and it is evident that they are the result of some powerful agency acting from within other than direct impact of the bullet. Here, no doubt, the "hydraulic theory" is sufficient to account for what is found; the enormous additional pressure suddenly exerted by its fluid contents in all directions on the inner surfaces of the closed vessel causes ruptures to occur at the points of least resistance, and this pressure expends itself in all directions. Naturally, therefore, it is found that ruptures are as likely to occur on the entrance side as on any other, whereas "explosive effects" on living tissues are almost confined to the exit side of the part struck. To account for these injuries by the aid of this "theory" is to overlook the simpler explanation and to adopt the more complex one. If the sudden increase of pressure on the liquids of the part penetrated by the bullet were the cause of the great disorganisation found in these cases it should be expected that the destruction would be most marked up and down the limb—that is, on either side of the direction of the bullet track; but quite different conditions are always found. "Explosive effects" are very seldom seen except where bone is traversed by the bullet. Delorme states that they are very exceptional where soft parts alone are implicated, and both cases are open to the same explanation. When a rifle bullet produces these so-called "explosive effects" on living tissues the track it makes is somewhat funnel-shaped, and that portion of its course from the aperture of entrance to the near side of the bone represents the tube of the funnel and from that point to the exit side represents the expanded portion of the funnel. In its passage through the soft parts, where the resistance it meets with is but slight, it makes a track apparently of less diameter than its own; but from the point at which the bone is fractured, and where real opposition is first met with, splinters and fragments of the harder substance are driven forwards with enormous energy acquired from the projectile, and proportional to the amount which the latter has lost, and these, as well as the particles of the soft tissues and even of the fluids themselves, act as secondary missiles on all the parts in their immediate vicinity, causing destruction in ever increasing severity until the exit side is reached, where it is greatest. Fragments of bone are driven violently forwards, and escape at the exit wound or through the other rents in the skin made by their passage; muscles and tendons, nerves and vessels protrude from the exit wound, which itself may be almost of any dimensions, whilst the seat of fracture is quite cleared of fragments, showing considerable loss of substance between the broken ends. Splinters are also often found embedded in the muscles and other soft tissues at some considerable distance. The destructive effects of the bullet are seen to have been expended in a direction mostly corresponding with its forward motion, not upwards and downwards, and on the entrance as well as on the exit side, as would have been the case had they been due to hydraulic pressure, and as is seen to occur in the experiment on the tin can filled with water. In a word, the so-called "explosive effects" seen in rifle-bullet wounds produced at short ranges are due directly to the projectile and indirectly to the action of the particles of the tissues themselves (bones, soft parts, and liquids), which are driven forwards with great velocity, and possessed, therefore, of great energy and destructive power. This view is held by Sir Thomas Longmore.[2] In like manner, Delorme states that too much importance has for many years been attached to the hydraulic theory in this connexion; it does account, he says, for extensive injuries in the hollow viscera (bladder, stomach, intestine, heart); here it is indisputable: "but in the case of solid organs filled with liquids (brain, spleen, liver, kidneys) it is not the sole cause, because, for these latter, it is necessary to have great regard for the effect of the solid particles projected forwards. For the other soft tissues it has no importance whatever."[3] Paul Bruns has directed his attention

[1] Dec. 29th, 1894, and Jan. 5th and 12th, 1895.

[2] Gunshot Injuries, p. 127.

[3] Chirurgie de Guerre, vol. ii., p. 996.